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**Issue Date: 10 April 2007**

**Case No.: 2004-BLA-6395**

**In the Matter of:**

**M. F. M., Widow,**
**Claimant**

**v.**

**Sewell Coal Co.,**
**Employer**

**And**

**Director, Office of Workers' Compensation Programs,**
**Party-In-Interest**

# DECISION AND ORDER AWARDING BENEFITS

This proceeding arises from a claim for benefits filed by M. F. M., the surviving spouse of R. L. M., a deceased coal miner, under the Black Lung Benefits Act, 30 U.S.C. §901, et seq. Regulations implementing the Act have been published by the Secretary of Labor in Title 20 of the Code of Federal Regulations.

Black lung benefits are awarded to coal miners who are totally disabled by pneumoconiosis caused by inhalation of harmful dust in the course of coal mine employment and to the surviving dependents of coal miners whose death was caused by pneumoconiosis. Coal workers' pneumoconiosis is commonly known as black lung disease.

A formal hearing was held before the undersigned on November 8, 2006 in Charleston, West Virginia. At that time, all parties were afforded full opportunity to present evidence and argument as provided in the Act and the regulations. The record consists of the hearing transcript, Director's Exhibits 1 through 31 (DX 1-31), Claimant's Exhibits (CX) 1, 2, 7 through 10, and Employer's Exhibits (EX) 2 through 7, and 9. The Claimant filed her brief on February 13, 2007; the Employer filed its brief on February 13, 2007; the Director did not file a brief.

The findings of fact and conclusions of law which follow are based upon my analysis of the entire record, including all documentary evidence admitted, arguments made, and the testimony presented.

## Procedural History

On May 12, 2003, the miner, R. L. M., passed away; on June 10, 2003, the Claimant, M. F. M., filed the current application for black lung benefits under the Act, as his surviving spouse (DX 3). On March 4, 2004, the District Director issued a Proposed Decision and Order awarding benefits (DX 18). Following the Employer's timely request for a formal hearing, this matter was referred to the Office of Administrative Law Judges on June 8, 2004 (DX 29).

The matter was scheduled for hearing before Administrative Law Judge Richard Morgan, but neither the Claimant nor her representative appeared. After Judge Morgan issued an order to show cause, counsel for the Claimant advised the Court that he had not received notification of the hearing. Judge Morgan subsequently continued the hearing, and the matter was assigned to Administrative Law Judge Michael Lesniak. The hearing that was scheduled for May 16, 2006 was subsequently continued to allow the Claimant time to have x-rays re-read. The matter was assigned to me, and I conducted a hearing on November 8, 2006 in Charleston, West Virginia.

## Issues

The following issues are contested by the Employer:

1. Whether Mr. M. had pneumoconiosis.
2. If so, whether his pneumoconiosis arose out of his coal mine employment.
3. Whether Mr. M.'s death was due to pneumoconiosis.

(DX 29; Tr. 17-19).

## Applicable Standard

The Regulations at 20 C.F.R. § 718 apply to survivors' claims which are filed on or after April 1, 1980. 20 C.F.R. § 718.1. Because the Claimant filed her survivor's claim after January 1, 1982, 20 C.F.R. § 718.205(c) applies to this claim.

The regulations provide that a survivor is entitled to benefits only where the miner died due to pneumoconiosis. 20 C.F.R. § 718.205(a). Mrs. M. must establish that: (1) Mr. M. was a coal miner; (2) Mr. M. suffered from pneumoconiosis at the time of his death; (3) Mr. M.'s pneumoconiosis arose out of his coal mine employment; and (4) Mr. M.'s death was caused by pneumoconiosis or pneumoconiosis was a substantially contributing cause or factor leading to his death. All elements of entitlement must be established by a preponderance of the evidence. *Strike v. Director, OWCP,* 817 F.2d 395, 399 (7th Cir. 1987). The survivor of a miner who was totally disabled due to pneumoconiosis at the time of death, but died due to an unrelated cause, is not entitled to benefits. 20 C.F.R. § 718.205(c). If the principal cause of death is a medical condition unrelated to pneumoconiosis, the survivor is not entitled to benefits unless the evidence

establishes that pneumoconiosis was a substantially contributing cause of the death. 20 C.F.R. § 718.205(c)(4).

The Board has held that death will be considered to be due to pneumoconiosis where the cause of death is significantly related to or significantly aggravated by pneumoconiosis. *Foreman v. Peabody Coal Co.,*8 B.L.R. 1-371 (1985). The United States Court of Appeals for the Sixth Circuit, in which the instant case arises, has held that pneumoconiosis is a substantially contributing cause of death if it hastens, even briefly, the miner's death. *See, Brown v. Rock Creek Mining Corp.,* 996 F.2d 812 (6th Cir. 1993)(J. Batchelder dissenting). *See also, Shuff v. Cedar Coal Co.,* 967 F.2d 977 (4th Cir. 1992), *cert. denied,* 113 S.Ct. 969 (1993); *Peabody Coal Co. V. Director, OWCP,* 972 F.2d 178 (7th Cir. 1992); *Lukosevicz v. Director, OWCP,* 888 F.2d 1001 (3rd. Cir. 1989).

The Board has held that in a Part 718 survivor's claim, the Judge must make a threshold determination as to the existence of pneumoconiosis under 20 C.F.R. § 718.202(a) before considering whether the miner's death was due to the disease under § 718.205. *Trumbo v. Reading Anthracite Co.,* 17 B.L.R. 1-85 (1993).

## Findings of Fact and Conclusions of Law

### *I. Background*

Mr. M. was born on June 2, 1920, and died on May 12, 2003 (DX 3). He and the Claimant, M. F. M., were married on August 16, 1939 (DX 1). The Director determined that Mrs. M. established that Mr. M. worked for 28 years as a coal miner. This determination is supported by Mr. M.'s Social Security earnings records, which also document that he last worked for Sewell Coal Company, for ten years ending in December 1982. I find that the Claimant has established 28 years of coal mine employment, and that Sewell Coal Company is properly designated as the responsible operator.[1]

Mrs. M. testified at the hearing. She stated that she and Mr. M. were married about 64 years at the time he passed away (Tr. 20). Mrs. M. testified that her husband worked for Sewell Coal from 1948 until about four years before his death (Tr. 21). He worked underground as a loader; for the last three years, he worked in the office outside as a dispatcher (Tr. 22). He had received a letter notifying him that he had changes in his lungs (Tr. 23). Mr. M. left the mines when they closed. At that time, he could not get his breath, and could not go up or down stairs, or walk very far. He used an inhaler and was on oxygen (Tr. 23). Mr. M. was treated for his lung condition by Dr. Diaz. He was in and out of the hospital for the last two or three years before he died (Tr. 24). According to Mrs. M., her husband never smoked (Tr. 25).

### *II. Medical Evidence*

[1] The Employer agrees that Mr. M. worked at least ten years as a coal miner, from 1972 through 1982 for Sewell Coal Company; it does not contest its status as the responsible operator (Tr. 18-19).

## X-Ray Evidence[2]

| *Exhibit No.* | *Date of X-Ray* | *Reading Date* | *Physician/ Qualifications* | *Impression* |
|---|---|---|---|---|
| DX 9 | 8-29-96 | 8-29-96 | Orbeta | Moderately severe interstitial fibrosis; no evidence of consolidation |
| DX 17 | 8-3-01 | 12-11-03 | Scott/B, BCR | Negative for pneumoconiosis |
| DX 9 | 8-3-01 | 8-3-01 | Younis | Diffuse changes of occupational lung disease; enlarging opacities in suprahilar regions, favoring conglomerate masses of pneumoconiosis |
| CX 8 | 8-3-01 | 9-1-06 | Cappiello/B, BCR | 2/2, p, q; Category A opacities |
| DX 9 | 9-4-01 | 9-4-01 | Leef | Increased densities in the upper lobes consistent with conglomerate occupational pneumoconiosis |
| DX 9 | 6-17-02 | 6-17-02 | Reifsteck | Diffuse bilateral interstitial changes; no new signs of consolidation |
| DX 17 | 8-15-02 | 12-11-03 | Scott/B, BCR | Negative for pneumoconiosis |
| DX 9 | 8-15-02 | 8-15-02 | Anton | Bilateral fibrotic changes in mid to upper zones bilaterally consistent with occupational pneumoconiosis |
| DX 8 | 8-16-02 | 8-16-02 | McJunkin | Extensive fibrotic changes bilaterally; improvement in density in left upper lung |
| DX 8 | 8-18-02 | 8-18-02 | Cordell | Interval improvement in right upper lobe density consistent with resolving atelectasis or pneumonia; chronic fibrotic changes both lungs |
| DX 9 | 9-16-02 | 9-16-02 | Smith | Coarse chronic appearing interstitial lung markings bilaterally consistent with chronic pulmonary fibrosis. |
| DX 17 | 9-16-02 | 12-11-03 | Wheeler/B, BCR | Negative for pneumoconiosis |
| DX 8 | 9-16-02 | 9-16-02 | Smith | Coarse chronic appearing interstitial lung markings bilaterally consistent |

[2] B - B Reader; and BCR - Board Certified Radiologist. These designations indicate qualifications a person may possess to interpret x-ray film. A "B Reader" has demonstrated proficiency in assessing and classifying chest x-ray evidence for pneumoconiosis by successful completion of an examination. A "Board Certified Radiologist" has been certified, after four years of study and an examination, as proficient in interpreting x-ray films of all kinds including images of the lungs.

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|---|---|---|---|---|
| | | | | with chronic pulmonary fibrosis; mild bilateral apical pleural thickening |
| CX 7 | 9-16-02 | 10-6-06 | Miller/B, BCR | 2/3, u, t; Category B opacities |
| DX 8 | 2-13-03 | 2-13-03 | Smith | Chronic appearing interstitial infiltrates predominantly in the perihilar and upper lobe regions bilaterally; bilateral apical pleural thickening; some chronic appearing interstitial changes in lung bases |
| DX 8 | 2-14-03 | 2-14-03 | Skeens | Increased interstitial lung markings bilaterally, predominantly involving the upper lung zones; superimposed acute infiltrate in left lung base probably representing pneumonitis |
| DX 8 | 2-17-03 | 2-17-03 | Anton | Persistent bilateral perihilar and upper lobe parenchymal changes which have appearance of occupational pneumoconiosis |
| DX 8 | 2-20-03 | 2-20-03 | Skeens | Extensive bilateral infiltrates, predominantly interstitial, consistent with chronic interstitial lung disease |
| DX 8 | 2-24-03 | 2-24-03 | Conner | No significant change in bilateral consolidation |
| DX 8 | 3-4-03 | 3-4-03 | Leef | Extensive infiltrates throughout both lungs, could represent conglomerate pneumoconiosis |
| EX 4 | 3-4-03 | 12-11-03 | Wheeler/B, BCR | Negative for pneumoconiosis |
| EX 4 | 3-4-03 | 12-11-03 | Scott/B, BCR | Negative for pneumoconiosis |
| CX 1 | 3-4-03 | 9-1-06 | Cappiello/B, BCR | 3/2, q, p; Category B opacities |
| CX2 | 3-4-03 | 10-6-06 | Miller/B, BCR | 3/2, u, e; Category B opacities |

## Medical Reports

*Dr. Hao Chang*

Dr. Chang treated Mr. M., and his handwritten notes are in the record (DX 9). They indicate that Dr. Chang treated Mr. M. for, among other things, severe COPD. Dr. Chang's records also include chest x-ray reports, which are included in the chart above.

*Richwood Area Community Hospital*

Mr. M. was brought to the Emergency Room on February 13, 2003 with complaints of congestion and shortness of breath. The reports note a history of pneumoconiosis and COPD. Mr. M. was admitted, with a diagnosis of left sided pneumonia and exacerbation of COPD; he was discharged on February 27, 2003.

Mr. M. visited the Emergency Room on August 15, 2002 with complaints of shortness of breath. His records note a history of pneumoconiosis.

The record also includes treatment notes from the hospital clinic, covering 2002 and 2003 (DX 7). On September 16, the examiner noted bibasilar crackles in Mr. M.'s chest. The impressions were COPD, pneumoconiosis, anxiety, and anemia. Dr. Farooq saw Mr. M. on September 30, 2002 noting a few basilar crackles in his chest, which was otherwise clear, with no wheezes, rhonchi or rub, but poor air movement, especially in the lower lobes. His diagnosis was chronic obstructive pulmonary disease, pneumoconiosis, anxiety, and iron deficient anemia. Dr. Farooq saw Mr. M. on October 28, 2002, noting a few bibasilar crackles in his chest, but no wheezes, rhonchi, or rubs, and poor air movement in the lower lobes. He diagnosed chronic obstructive pulmonary disease, pneumoconiosis, anxiety, and iron deficiency anemia. Dr. Farooq next saw Mr. M. on January 30, 2003, noting a few basilar crackles in his chest, which was otherwise clear. He diagnosed anorexia, COPD, pneumoconiosis, anxiety, and iron deficiency anemia. Dr. Mudassir Nawaz treated Mr. M. on April 1, 2003, noting that he had a history of COPD. On examination, Mr. M.'s chest was clear to auscultation and percussion. Dr. Nawaz's assessment was COPD/pneumoconiosis; anxiety; GERD with duodenitis; and mild renal insufficiency.

The clinic records include a "Death Note" by Dr. Farooq, dated May 12, 2003. Dr. Farooq indicated that he had received a call from the paramedic who treated Mr. M., who told Dr. Farooq that Mr. M. died in his bed. Dr. Farooq completed Mr. M.'s death certificate, recording chronic obstructive pulmonary disease as the immediate cause of death, with pneumoconiosis and anemia as the underlying causes (DX 6).

*Dr. Kirk Hippensteel*

Dr. Hippensteel reviewed Mr. M.'s medical records at the Employer's request, and prepared a report dated December 6, 2004 (EX 2). Dr. Hippensteel stated that Mr. M. had chronic interstitial changes on his chest x-ray that were thought by some to represent pneumoconiosis, although several B readers felt that the fact that there were no rounded opacities argued against coal workers' pneumoconiosis. He noted that the available pulmonary function studies showed no significant ventilatory abnormality; there were variable gas exchange abnormalities, with acute exacerbations of chronic obstructive pulmonary disease. Mr. M. also had a history of cardiac arrhythmia, and he died at home, which Dr. Hippensteel felt made it likely that the cardiac arrhythmia was the cause of his death, despite the fact that Dr. Farooq did not note it on the death certificate.

According to Dr. Hippensteel, the data available to him, "overall," did not show that Mr. M. had coal workers' pneumoconiosis, or a significant impairment from a pulmonary standpoint from any cause. Rather, he had acute exacerbations of breathing problems, which were not

reflective of permanent impairment. Although the exact cause of his death was not determined, the fact that Mr. M. experienced a quick death at home favored cardiac arrhythmia as the cause. Dr. Hippensteel stated that such arrhythmias would relate to Mr. M.'s heart disease, but not his lung disease, and therefore were not referable to his previous coal mine dust exposure.

Dr. Hippensteel concluded that the functional evidence did not show a permanent level of impairment from a pulmonary standpoint, which would have kept Mr. M. from returning to his previous job in the mines, even if it were stipulated that he had coal workers' pneumoconiosis. Mr. M. had other medical problems, such as his heart arrhythmia, that appeared to keep him from going back to work in the mines, and also caused his death.

Dr. Hippensteel testified by deposition on September 27, 2005 (EX 6). He compared the results of pulmonary function studies performed in 1980, which were borderline normal, to studies done on April 16, 2003, which showed severe air flow obstruction. At this time, Mr. M. had been hospitalized for pneumonia, and Dr. Hippensteel felt that he would have some acute effects tied in with that. The studies suggested that Mr. M. had developed severe obstructive disease, just after recovering from an acute and severe infection; he did not have obstructive disease in 1980.

Dr. Hippensteel described tuberculosis and sarcoidosis as two types of granulomatous disease that can create conglomerate opacities in the upper lobes of the lungs, without significant impairment in function. In contrast, coal workers' pneumoconiosis is less localized, and is also called progressive massive fibrosis, which is usually tied in with more changes in function referable to the progression of fibrosis in the lungs. He stated that interstitial fibrosis is a nonspecific term, and it can occur with causes, including chronic infections, which leave scars behind. It can include diseases that are idiopathic, and that make interstitial fibrosis come on without any association with any occupation or illness. At times, it can be associated with changes referable to coal workers' pneumoconiosis.

Dr. Hippensteel noted that the interstitial infiltrates that some physicians saw on x-rays were mostly in Mr. M.'s lower lobes and mid lung zones, and were not associated with the fibrotic pattern that they found in his upper lobes. The physicians did not see coalescing nodules in the upper lungs consistent with pneumoconiosis as the cause for these changes; rather, they saw the interstitial changes in areas separate from the conglomerate opacities, which was not indicative of coal workers' pneumoconiosis.

Dr. Hippensteel pointed out that Mr. M. had been hospitalized just before his death with pneumonia associated with a lot of dyspnea. He had also been found to have frequent premature ventricular contractions and an arrhythmia, and he was also anemic, with diagnoses that included left ventricular hypertrophy, hyperthyroidism, allergic rhinitis, and skin cancer. According to Dr. Hippensteel, some people with allergic rhinitis also have allergies that affect their lower airway, even though no specific diagnosis of asthma was made. This would cause an obstructive disease that would result in intermittent exacerbations such as Mr. M. had, which required intermittent therapy. In contrast, exacerbations would not be expected with pneumoconiosis.

Dr. Hippensteel stated that it was not clear as to the exact cause of Mr. M.'s death, as he died at home, without any observation that it was associated with an exacerbation of breathing. That lent credence to his feeling that arrhythmia was the most likely cause, because it develops quickly, does not produce preceding symptoms, and would result in a quick passing at home, without the symptoms of exacerbation of breathing that would precipitate a trip to the emergency room. But he did not think that there was any indication in the records that coal workers' pneumoconiosis or coal dust exposure had any impact on his death.

According to Dr. Hippensteel, Mr. M.'s arrhythmia first showed up in 1996. He testified that the most common cause of arrhythmia is heart disease, especially coronary artery disease. He acknowledged that Mr. M. did not have evidence of a heart attack, or coronary artery disease. He did not have a complete workup for coronary artery disease. But according to Dr. Hippensteel, arrhythmias occur even in the absence of coronary artery disease; they can be a primary heart problem without associated coronary artery disease. He described an arrhythmia as an irregular heartbeat. Those that are less serious cause symptoms of weakness and light headedness, and can be controlled with medication. There are also more serious types that can cause sudden death. According to Dr. Hippensteel, Mr. M. had the ventricular type of arrhythmia, which is the more serious type.

*Dr. Samuel V. Spagnolo*

Dr. Spagnolo reviewed Mr. M.'s medical records at the Employer's request, and prepared a report dated November 15, 2004 (EX 3). Dr. Spagnolo concluded that Mr. M. had sufficient exposure to coal dust to result in pneumoconiosis, but based on the clinical, laboratory, and radiographic evidence, he did not believe that there was sufficient objective medical evidence to indicate that he had a respiratory or pulmonary impairment or condition that was contributed to or aggravated by the inhalation of coal dust. Nor did Dr. Spagnolo believe that there was sufficient evidence of complicated pneumoconiosis or progressive massive fibrosis caused by the inhalation of coal dust.

According to Dr. Spagnolo, there is no evidence that Mr. M. had any obstructive or restrictive lung impairment before he left his coal mine employment. His arterial blood gas study results in 2002 did not support the presence of any chronic progressive lung disease that would have prevented him from performing his previous coal mining work. His lung examinations throughout the 1990s were reported as normal on most occasions, and he was doing well, with no complaints of breathing difficulties or chest pain. In 2002, Mr. M. began having recurrent episodes of acute pneumonia.

Dr. Spagnolo placed the greatest weight on the chest x-ray reports by Dr. Wheeler, Dr. Scatarige, and Dr. Scott, because they are university based radiologists with outstanding credentials in the evaluation of x-rays of persons with occupational exposure and related lung disease. He noted that their reports were thoughtful and complete, and they felt that the x-rays did not support a diagnosis of pneumoconiosis. According to Dr. Spagnolo, based on the clinical and radiological evidence, it was likely that Mr. M. was having acute episodes of recurrent aspiration, leading to recurring pneumonia, in his later years.

Dr. Spagnolo noted that Mr. M. died suddenly at age 82, many years after he left coal mine employment, from multiple medical problems, including gastroesophageal reflux, recurrent pneumonia, duodenitis, cardiac and renal disease (arrhythmia), prostate enlargement, and anemia. But none of these conditions is related to his coal dust exposure or coal mine employment. In Dr. Spagnolo's opinion, none of Mr. M.'s symptoms, complaints, or medical conditions before his death were related to his coal dust exposure or coal mine employment. His death was unrelated to and not hastened, even briefly, by pneumoconiosis, nor was pneumoconiosis a contributing factor in his death.

Dr. Spagnolo testified by deposition on September 21, 2005 (EX 5). He described "infiltrate" as a term that refers to an irregular density on x-ray, which is not necessarily specific for any particular lung disease. In contrast, pneumoconiosis is classically described as being very small round densities. He also stated that pleural thickening is not caused by coal workers' pneumoconiosis. The most common cause is an old infection, or inflammatory process. He noted that starting in early 2002, Mr. M. had multiple pneumonias. More importantly, his father had tuberculosis, and that there is a possibility that he could have passed it to Mr. M.

Dr. Spagnolo felt that Mr. M. had significant risk factors for developing pneumonia. It was consistently noted that he had GERD, as well as duodenitis, which are very common causes of aspiration pneumonia, particularly in the elderly. He felt that this was the most reasonable explanation for Mr. M.'s recurrent pneumonias. But he was not aware of any medical evidence that coal workers' pneumoconiosis or coal dust exposure predispose a person to a greater risk of pneumonia.

Dr. Spagnolo noted that, starting in 2002, Mr. M.'s arterial blood gas studies were variable, which was more consistent with a person having acute changes, including pneumonia, which resolve. In contrast, pneumoconiosis causes a consistent impairment.

Addressing Dr. Guadiano's diagnosis of progressive massive fibrosis or complicated pneumoconiosis, Dr. Spagnolo noted that there was no report of any large category opacities in the x-ray reports he reviewed.

Dr. Spagnolo felt that Mr. M. had multiple recurrent pneumonias, significant heart disease, significant gastroesophageal reflux disease, anemia, and renal disease, and in the last year or two of his life he worsened due to these conditions. He stated that Mr. M. probably had a cardiac arrhythmia, irregular heart rate, or acute myocardial infarction. In view of his recurrent pneumonias, he may have had a massive aspiration pneumonia. But none of these conditions was related to pneumoconiosis or coal dust exposure.

*Dr. Paul S. Wheeler*

Dr. Wheeler reviewed the x-ray reports by Dr. Cappiello and Dr. Miller, and wrote a report dated October 30, 2006 (EX 7). He stated that he found their reports inconsistent and confusing. With respect to Dr. Cappiello's interpretation of the August 3, 2001 x-ray, he noted that Dr. Cappiello indicated in Section 3B of the ILO form that there were right pleural plaques, which, according to Dr. Hippensteel, are specific for asbestos exposure. However, in the

narrative, Dr. Cappiello contradicted this by noting right chest wall pleural thickening, which is a nonspecific pleural fibrosis. Similarly, with the September 16, 2002 x-ray, Dr. Cappiello noted pleural plaques in the form, but in the narrative reported pleural thickening. Finally, on the March 4, 2003 x-ray, Dr. Cappiello reported bilateral pleural plaques on the ILO form, but reported bilateral chest wall pleural thickening in the narrative, which, according to Dr. Wheeler, is a contradiction and quality discrepancy.

Dr. Wheeler noted that Dr. Cappiello reported small round opacities, and "A" or "B" large opacities, indicating that he believed the x-rays showed complicated pneumoconiosis, since asbestos exposure does not cause large opacities. But he reported pleural plaques on section 3B, which means asbestos exposure; he then contradicted this by noting pleural thickening in the narrative portions. According to Dr. Wheeler, pleural fibrosis is nonspecific, and caused by inflammatory disease, cancer, and trauma. Dr. Wheeler stated: "His reports indicate mixed pneumoconiosis which may occur in other countries but is now rare in America." Dr. Wheeler was not able to see the "p" or "q" small round nodules that Dr. Cappiello reported.

With respect to Dr. Miller's interpretations of the August 3, 2001 and September 16, 2002 x-rays, Dr. Wheeler noted that he reported right pleural plaques in the ILO form, but right pleural thickening in the narrative, which, according to Dr. Wheeler, is a contradiction, because pleural plaques are specific for asbestos exposure, and pleural thickening is nonspecific. In his review of the March 4, 2003 x-ray, Dr. Miller also reported bilateral pleural plaques on the form, and bilateral pleural thickening in the narrative section.

According to Dr. Wheeler, Dr. Miller's notation of "u" and "t" small opacities indicates advanced asbestosis with large opacities. But only coal workers' pneumoconiosis or silicosis causes large opacities. He stated that Dr. Miller did not mention small round opacities, which are the building blocks that merge to form large opacities of coal workers' pneumoconiosis. He stated that the asbestos exposures in America since World War II have typically caused benign asbestos-related pleural plaques, which are collagen deposits in the parietal pleura, and which calcify after several decades, making them quite obvious on routine chest x-rays. But there were no benign asbestos-related pleural plaques on the examinations, only nonspecific pleural fibrosis. In Dr. Wheeler's opinion, the pleural thickening was nonspecific, and due to healed granulomatous disease.

According to Dr. Wheeler,

> The ILO-Guidelines are very clear that <u>there is no radiographic abnormality that is diagnostic of any pneumoconiosis</u> but there are certain patterns that are consistent with various pneumoconioses. This is important because several other diseases can cause small round nodules including various granulomatous diseases and metastases, while many diseases are capable of causing small patchy and irregular opacities with or without pleural involvement. The "OTHER COMMENTS" Section in the ILO-form allows B-readers to express uncertainties and to suggest other possibilities for abnormal x-ray patterns. Dr. Miller and Dr. Cappiello do not mention other conditions in that Section which could cause the lung opacities some of which are serious.

Dr. Wheeler felt that another peculiar feature of these reports was their indication of involvement in six lung zones. He stated that he had been a B reader for three decades, and had only seen six zone lung involvement among older miners, in the large surveys done for the government in the 1970s. According to Dr. Wheeler, typically, pneumoconiosis and silicosis involve the mid and upper lung zones, and asbestosis involves the lower lung zones, and occasionally the mid and peripheral upper lung zones. But involvement of six lung zones now by any pneumoconiosis is very rare.

Dr. Wheeler insisted that, regardless of the x-ray findings, in any case with significant lung disease, an exact diagnosis is needed for proper therapy. This usually requires open lung biopsy, transbronchial biopsy, or convincing microbiology. He stated that the ILO-classification system was never intended to be used for making clinical judgments, especially when active inflammatory disease or cancer could be the cause of round nodules or linear, irregular or patchy small opacities.

Dr. Wheeler felt that the extensive infiltrates on the chest x-rays represented inflammatory disease, such as histoplasmosis, which is endemic in large parts of the eastern United States, and which is capable of causing the infiltrates, fibrosis, and pleural disease. He found no small nodules in the mid and upper lungs to suggest pneumoconiosis, and there were no calcified benign asbestos-related pleural plaques, which should be present with pulmonary asbestosis. He stated that, "of course," pneumoconiosis does not involve the pleura, and only pneumoconiosis and silicosis are capable of causing large opacities, which he did not see on the x-rays.

According to Dr. Wheeler, the reports by Dr. Miller and Dr. Cappiello are inconsistent: both report complicated pneumoconiosis with large opacities, while only one reported small round nodules that could merge to cause the large opacities. Both reported pleural plaques, which are specific for asbestos, but also stated "pleural thickening," which is nonspecific, and produced by a variety of diseases from trauma to inflammation and cancer.

*Dr. William W. Scott*

Dr. Scott also addressed the x-ray interpretations by Dr. Cappiello and Dr. Miller in a report dated November 2, 2006 (EX 9). In his review of the x-rays performed on August 3, 2001, September 16, 2002, and March 4, 2003, Dr. Scott noted a combination of linear interstitial fibrosis, and patchy areas of alveolar infiltrate, predominantly in the upper lung zones. There was thickened pleura in the upper right lung. Dr. Scott felt that these changes probably represented nonspecific linear interstitial fibrosis, and an inflammatory process such as tuberculosis of unknown activity. He indicated that there was no dramatic progression of disease, but in the presence of areas of alveolar infiltrate, active tuberculosis could not be excluded. According to Dr. Scott, fungal disease and other granulomatous disease might give a similar pattern.

Dr. Scott did not feel that the x-ray findings represented silicosis or pneumoconiosis, because there was no background of small rounded opacities. There was also considerable pleural involvement, especially in the right upper chest, which was much more suggestive of

tuberculosis, but was not typical for silicosis or pneumoconiosis. According to Dr. Scott, as there was no background of small rounded opacities compatible with silicosis or pneumoconiosis, it would be unreasonable to claim that the areas of alveolar opacification represent large opacities of silicosis or pneumoconiosis.

According to Dr. Scott, the notations of indistinct heart border, indistinct hemidiaphragm, and honeycombing by Dr. Cappiello on his report of the March 4, 2003 x-ray are features of asbestosis and UIP, but not of silicosis or pneumoconiosis. Nor is pleural thickening a feature of silicosis or pneumoconiosis. It can be a feature of asbestos exposure, but it is usually bilateral, and tends to calcify after a period of time. Unilateral pleural thickening is usually post-infectious. Dr. Scott stated that "Drs. Cappiello and Miller seem confused about the appearance of silicosis/CWP."

## DISCUSSION

*Existence of Pneumoconiosis*

Pneumoconiosis is defined, by regulation, as a "chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment." 20 C.F.R. § 718.201. The regulations at 20 C.F.R. § 718.203(b) provide that, if it is determined that the miner suffered from pneumoconiosis and engaged in coal mine employment for ten years or more, there is a rebuttable presumption that the pneumoconiosis arose out of such employment. If, however, it is established that the miner suffered from pneumoconiosis but worked less than ten years in the coal mines, then the claimant must establish causation by competent evidence. *Stark v. Director, OWCP,*9 B.L.R. 1-36 (1986); *Hucker v. Consolidation Coal Co.,*9 B.L.R. 1-137 (1986). The Board has held that the burden of proof is met under § 718.203(c) where "competent evidence establish(es) that his pneumoconiosis is significantly related to or substantially aggravated by the dust exposure of his coal mine employment." *Shoup v. Director, OWCP,* 11 B.L.R. 1-1101-112 (1987). Specifically, the record must contain *medical* evidence to demonstrate causation. *Baumgartner v. Director, OWCP,* 9 B.L.R. 1-65, 1-66 (1986)(administrative law judge cannot infer causation based solely upon claimant's employment history); *Tucker v. Director, OWCP,* 10 B.L.R. 1-35, 1-39 (1987)(it was error for the administrative law judge to rely solely upon lay testimony to find causation established).

The existence of pneumoconiosis may be established by any one or more of the following methods: (1) chest x-rays; (2) autopsy or biopsy; (3) by operation of presumption; or (4) by a physician exercising sound medical judgment based on objective medical evidence. 20 C.F.R. § 718.202(a). In this case, there are a number of narrative x-ray reports that were done while Mr. M. was hospitalized. In addition, the parties have submitted ILO readings of several of these x-rays.

As early as August 29, 1996, Dr. Orbeta noted moderately severe interstitial fibrosis, with no evidence of consolidation on Mr. M.'s x-ray. The next x-ray of record was done on August 3, 2001, and Dr. Younis read it to show diffuse changes of occupational lung disease, and enlarging opacities in the suprahilar regions, favoring conglomerate masses of pneumoconiosis. Dr.

Cappiello, who is dually qualified, reviewed this x-ray and reported pneumoconiosis 2/2, with category A opacities. However, Dr. Scott, who is also dually qualified, found this x-ray to be negative for pneumoconiosis. He did indicate that he saw bilateral diffuse interstitial and alveolar infiltrates, possibly tuberculosis superimposed on non-specific linear fibrosis.

Dr. Leef reviewed Mr. M.'s September 4, 2001 x-ray, and described increased densities in the upper lobes, consistent with conglomerate occupational pneumoconiosis. On the June 17, 2002 x-ray, Dr. Riefsteck noted diffuse bilateral interstitial changes, with no new signs of consolidation.

On August 15, 2002, Mr. M. underwent an x-ray, which Dr. Anton reported showed bilateral fibrotic changes in the mid to upper zones bilaterally, consistent with occupational pneumoconiosis. However, Dr. Scott reviewed this film, which he found to be negative for pneumoconiosis. Mr. M. had two more x-rays during this hospitalization. On August 16, 2002, Dr. McJunkin interpreted his x-ray as showing extensive fibrotic changes bilaterally, and improvement in the density in the left upper lung. Two days later, on August 18, Dr. Cordell noted interval improvement in the right upper lobe density, consistent with resolving atelectasis or pneumonia, and chronic fibrotic changes in both lungs.

An x-ray performed on September 16, 2002 was interpreted by Dr. Smith to show coarse and chronic appearing interstitial lung markings bilaterally, consistent with chronic pulmonary fibrosis. Dr. Miller, who is dually qualified, read this x-ray, noting pneumoconiosis 2/3, and category B opacities. Dr. Wheeler, however, read this x-ray as negative for pneumoconiosis.

Mr. M.'s x-ray of February 13, 2003 was interpreted by Dr. Smith to show chronic appearing interstitial infiltrates, predominantly in the perihilar and upper lobe regions bilaterally, bilateral apical pleural thickening, and some chronic appearing interstitial changes in the lung bases. An x-ray done the following day, on February 14, was read by Dr. Skeens to show increased interstitial lung markings bilaterally, predominantly involving the upper lung zones, and superimposed acute infiltrate in the left lung base that probably represented pneumonitis.

When Mr. M's x-ray was taken on February 17, 2003, Dr. Anton found persistent bilateral perihilar and upper lobe parenchymal changes that had the appearance of occupational pneumoconiosis. Three days later, on February 20, Dr. Skeens reviewed Mr. M.'s x-ray, noting extensive bilateral infiltrates, predominantly interstitial, consistent with chronic interstitial lung disease. On February 24, Dr. Conner noted no significant change in the bilateral consolidation.

The last x-ray, performed on March 4, 2003, was read by Dr. Leef, who noted extensive infiltrates throughout both lungs, that could represent conglomerate pneumoconiosis. Dr. Cappiello and Dr. Miller, both dually qualified, found pneumoconiosis 3/2, with category B opacities, while Dr. Scott and Dr. Wheeler, also both dually qualified, found this film to be negative for pneumoconiosis.

Thus, the ILO interpretations by dually qualified physicians are equally balanced between readings that are positive and negative for pneumoconiosis. Although the radiologists who read Mr. M.'s x-rays while he was in the hospital consistently found abnormalities consistent with

pneumoconiosis, the qualifications of these radiologists are not in the record, and thus their interpretations are not sufficient to tip the scales in favor of a finding of pneumoconiosis. I find that the x-ray evidence is essentially in equipoise, and thus Mrs. M. has not established the existence of pneumoconiosis by a preponderance of the x-ray evidence.

There is no autopsy or biopsy evidence in the record, and thus Mrs. M. has not established that Mr. M. had pneumoconiosis by these means.

Under § 718.202(a)(4), Mrs. M. can also establish that Mr. M. suffered from pneumoconiosis by well-reasoned, well-documented medical reports. A "documented" opinion is one that sets forth the clinical findings, observations, facts and other data on which the physician based the diagnosis. *Fields v. Island Creek Coal Co.,* 10 B.L.R. 1-19 (1987). An opinion may be adequately documented if it is based on items such as a physical examination, symptoms, and the patient's history. *See,* 20 C.F.R. § 718.107, *Hoffman v. B&G Construction Co.,* 8 B.L.R. 1-65 (1985); *Hess v. Clinchfield Coal Co.,* 7 B.L.R. 1-295 (1984). A report which is better supported by the objective medical evidence of record may be accorded greater probative value. *Minnich v. Pagnotti Enterprises, Inc.,* 9 B.L.R. 1-89, 1-90 n.1 (1986); *Wetzel v. Director, OWCP,*8 B.L.R. 1-139 (1985).

A "reasoned" opinion is one in which the administrative law judge finds the underlying documentation adequate to support the physician's conclusions. *Fields, supra.* Indeed, whether a medical report is sufficiently documented and reasoned is for the administrative law judge as the finder of fact to decide. *Clark v. Karst-Robbins Coal Co.,* 12 B.L.R. 1-149 (1989)(en banc). Moreover, statutory pneumoconiosis is established by well-reasoned medical reports which support a finding that the miner's pulmonary or respiratory condition is significantly related to or substantially aggravated by coal dust exposure. *Wilburn v. Director, OWCP,* 11 B.L.R. 1-135 (1988). An equivocal opinion, however, may be given little weight. *Justice v. Island Creek Coal Co.,* 11 B.L.R. 1-91 (1988); *Snorton v. Zeigler Coal Co.,* 9 B.L.R. 1-106 (1986).

Dr. Chang treated Mr. M. for a number of conditions over the years before his death, including severe COPD. However, his records do not include any evaluation for, or diagnosis of pneumoconiosis.

Mr. M. was also treated at the Richwood Area Community Hospital, as well as the Clinic. Mr. M. was seen at the Clinic five times between September 2002 and January 2003. He was diagnosed with COPD, pneumoconiosis, anxiety, anemia, GERD with duodenitis, and mild renal insufficiency. However, there is no indication that any objective testing was done, nor is the basis for the diagnosis of pneumoconiosis stated.

Mr. M. visited the Richwood Area Community Hospital emergency room on August 15, 2002, and again on February 13, 2003. On both occasions, his records included a notation of a history of pneumoconiosis. But there are not objective findings or test results to support a finding of pneumoconiosis, or any evaluation, other than the notation of the history of pneumoconiosis.

Dr. Hippensteel, who reviewed medical records at the Employer's request, concluded that this evidence, overall, did not show that Mr. M. had coal workers' pneumoconiosis; rather, he

had acute exacerbations of breathing problems, not reflective of permanent impairment.[3] Dr. Hippensteel did not review x-ray films himself, but discussed the interpretations of other readers. Dr. Hippensteel's report and deposition testimony are confusing: although he stated that the data available to him "overall" did not show that Mr. M. had coal workers' pneumoconiosis, his comments and opinions are addressed to the question of whether Mr. M. suffered from a pulmonary impairment, the nature of the conglomerate opacities in Mr. M.'s upper lobes, and the cause of his death. He did not have available the positive interpretations by Dr. Miller and Dr. Cappiello, who are both dually qualified interpreters.

Dr. Spagnolo also reviewed medical records at the Employer's request, and concluded that there was not sufficient objective medical evidence to indicate a respiratory or pulmonary impairment or condition related to the inhalation of coal mine dust. He relied on the x-ray reports by Dr. Wheeler, Dr. Scott, and Dr. Scatarige, because they are university based radiologists with outstanding credentials in the evaluation of x-rays of persons with occupational exposure and related lung disease. But he did not have the opportunity to review the interpretations of Dr. Cappiello and Dr. Miller, who are also dually qualified and well credentialed radiologists. Dr. Spagnolo also relied on the fact that Mr. M. did not have a pulmonary impairment before he left the coal mines, and his lung examinations throughout the 1990s were reported as normal on "most" occasions.[4] Dr. Spagnolo claimed that Mr. M.'s arterial blood gas study results in 2002 did not support the presence of a chronic progressive lung disease; but he did not address the fact that Dr. Chang's treatment notes document severe and worsening COPD after the "1990s." Nor is it relevant that Mr. M. began having recurrent episodes of acute pneumonia in 2002. I find that Dr. Spagnolo's opinions are not reliable, as they are based on an incomplete and selective review of the available medical evidence.

Dr. Wheeler addressed the x-ray interpretations by Dr. Cappiello and Dr. Miller, taking issue with their designation of pleural plaques on the form, but pleural thickening in the narrative, which he felt was inconsistent. He did not adequately explain why these designations were inconsistent, or why such findings could not co-exist. Nor did he indicate why this was relevant to their findings of pneumoconiosis. He also discussed what he felt was a "peculiar feature" of these reports, that is, their indication of involvement in six lung zones. According to Dr. Wheeler, pneumoconiosis and silicosis "typically" involve the mid and upper zones, while asbestosis involves the lower, and occasionally, mid lung zones. He did not offer support for this conclusion, or indicate any basis for a finding of "asbestosis." Dr. Wheeler also made it clear that he feels that x-rays are not sufficient for diagnosis of a significant lung disease, and usually open lung biopsy, transbronchial biopsy, or convincing microbiology is necessary. In his opinion, the ILO classification system was never intended to be used for making clinical judgments. However, the statute does not require a "clinical" judgment in order for a miner to be entitled to benefits, and a miner is not required to confirm x-ray findings by invasive procedures to obtain tissue confirmation before he is entitled to benefits.

[3] This is at odds with Dr. Chang's office notes, which reflect that Mr. M. suffered from severe and progressively worsening COPD.

[4] Dr. Spagnolo's opinions appear to ignore the concept that pneumoconiosis is a progressive condition that can worsen after exposure to coal mine dust ceases.

Dr. Scott also discussed the interpretations by Dr. Cappiello and Dr. Miller. Dr. Scott did not feel that the x-rays showed silicosis or pneumoconiosis, because there was no background of small rounded opacities. Clearly, he disagreed with the interpretations by Dr. Cappiello and Dr. Miller, who did find a background of small rounded opacities, as shown by their designation of a profusion of small opacities.

However, other than the notations of a diagnosis of or history of pneumoconiosis in Mr. M.'s hospital record, which are unsupported by objective clinical or testing findings, there is no physician who has concluded that Mr. M. had pneumoconiosis. Accordingly, I find that Mrs. M. has not established that Mr. M. had pneumoconiosis by virtue of the medical opinion evidence.

Section 718.202(a)(3) provides that pneumoconiosis may be established if any one of several cited presumptions are found applicable. In the instant case, the presumption of § 718.305 does not apply to claims filed after January 1, 1982. Section 718.306 does not apply to claims where the miner died after March 1, 1978. Section 718.304 allows a presumption of complicated pneumoconiosis where, *inter alia*, an x-ray "yields one or more large opacities (greater than 1 centimeter in diameter) and would be classified in Category A, B, or C" if such miner is suffering or suffered from a chronic dust disease of the lung. 20 C.F.R. § 718.304(a). However, if the employer can affirmatively show the opacity is something other than pneumoconiosis, the x-ray loses force, and the claimant loses the benefit of the presumption. *See Eastern Associated Coal Corp. v. Director, OWCP [Scarbro]*, 220 F.3d 250, 256 (4th Cir. 2000).

In *Eastern Associated Coal Corporation v. Director, OWCP, supra,* the Fourth Circuit discussed the three different ways set forth in the statute to establish the existence of statutory complicated pneumoconiosis in order to invoke the irrebuttable presumption at § 718.304, and noted that in applying the standards set forth in each prong,

> [O]ne must perform equivalency determinations to make certain that regardless of which diagnostic technique is used, the same underlying condition triggers the irrebuttable presumption.

*Id.* at 255, 256, *citing Double B Mining, Inc., v. Blankenship,* 177 F.3d 240, 243 (4th Cir. 1999). Additionally, the Court stated that

> "[B]ecause prong (A) sets out an entirely objective scientific standard" –i.e. an opacity on an x-ray greater than one centimeter –x-ray evidence provides the benchmark for determining what under prong (B) is a "massive lesion" and what under prong (C) is an equivalent diagnostic result reached by other means.

*Id. at 256, citing Double B Mining* at 243.

Although the Court acknowledged that a finding of statutory complicated pneumoconiosis may be based on evidence presented under a single prong, the Court also noted that the ALJ must review the evidence under each prong for which relevant evidence is presented, to determine if complicated pneumoconiosis is present. The Court stated that:

> Evidence under one prong can diminish the probative force of evidence under another prong if the two forms of evidence conflict. Yet, "a single piece of relevant evidence," *Lester [Lester v. Director, OWCP], 993 F.2d at 1145,* can support an ALJ's finding that the irrebuttable presumption was successfully invoked if that piece of evidence outweighs conflicting evidence in the record.

*Id.*

As the Court noted, even if there is some x-ray evidence that indicates that there are opacities that would satisfy the requirements of prong (A), if there is other x-ray evidence available, or other evidence relevant to an analysis under prongs (B) or (C), then all of the evidence must be considered to determine whether the evidence as a whole indicates a condition of such severity that it would produce opacities greater than one centimeter in diameter on an x-ray. The Court stated:

> Of course, if the x-ray evidence vividly displays opacities exceeding one centimeter, its probative force is not reduced because the evidence under some other prong is inconclusive or less vivid. Instead, the x-ray evidence can lose force **only if other evidence affirmatively shows** that the opacities are not there or are not what they seem to be, perhaps because of an intervening pathology, some technical problem with the equipment used, or incompetence of the reader.

*Id.* (emphasis added).

*1. Existence of an Opacity Greater than 1 Centimeter*

In this case, there is x-ray evidence that there are opacities that would satisfy the requirements of prong (A), in the form of the interpretations by Dr. Miller and Dr. Cappiello. However, there is also other x-ray evidence available, and thus all of the evidence must be considered to determine whether the evidence as a whole indicates a condition of such severity that it would produce opacities greater than one centimeter in diameter on an x-ray.

Mr. M. underwent an x-ray on August 3, 2001, which was interpreted by the attending radiologist, Dr. Younis, as showing diffuse changes of occupational lung disease, and enlarging opacities in the suprahilar regions that favored conglomerate masses of pneumoconiosis. Dr. Cappiello, a dually qualified interpreter, who interpreted this x-ray and completed an ILO form, categorized it as showing pneumoconiosis 2/2, with category A opacities. However, Dr. Scott, who is also dually qualified, interpreted this x-ray as negative for pneumoconiosis. In the narrative portion of his report, Dr. Scott described bilateral diffuse interstitial and alveolar infiltrates, possibly tuberculosis superimposed on non-specific linear fibrosis.

Mr. M. underwent an x-ray on September 16, 2002, which was interpreted by the attending radiologist, Dr. Smith, as showing bilateral coarse chronic appearing interstitial lung markings, consistent with chronic pulmonary fibrosis, and mild bilateral apical pleural thickening. Dr. Miller, who is dually qualified, read Mr. M.'s September 16, 2002 x-ray as showing pneumoconiosis 2/3, with category B opacities. On the other hand, Dr. Wheeler, who is

dually qualified, interpreted this x-ray as negative for pneumoconiosis. In the narrative portion of his report, Dr. Wheeler noted moderate coarse infiltrates or fibrosis in the upper lobe, compatible with inflammatory disease more likely than cancer, and minimal right apical pleural thickening or possible small loculated pleural effusion in the upper right apex.

Mr. M. underwent x-ray examination on March 4, 2003, and the attending radiologist, Dr. Leef, reported extensive infiltrates throughout both lungs that could represent conglomerate pneumoconiosis. Dr. Cappiello and Dr. Miller both noted pneumoconiosis 3/2, with category B opacities, while Dr. Wheeler and Dr. Scott both found the film to be negative for pneumoconiosis. Again, Dr. Wheeler described moderate coarse infiltrates or fibrosis in the upper lobes compatible with inflammatory disease more likely than cancer. In his narrative, Dr. Scott reported bilateral infiltrates with alveolar and interstitial components and a pleural reaction in the lateral right upper lung, which could be tuberculosis superimposed on prior non-specific fibrosis.

Many of the narrative x-ray interpretations describe the presence of enlarging opacities, increased densities, and extensive infiltrates that could represent conglomerate pneumoconiosis, which many of the physicians attributed to conglomerate occupational pneumoconiosis. None of these interpretations, however, address the question of whether these densities or masses would appear on x-ray as Category A, B, or C opacities. While these narrative interpretations do not fall in any of the categories of evidence specifically set out in the statute, however, they certainly do not detract from a conclusion that there are large opacities on Mr. M.'s x-rays, as reported by Dr. Miller and Dr. Cappiello, or that Mr. M.'s condition was of such severity that it would produce such large opacities on x-ray. Nor do these narrative interpretations provide affirmative evidence that the large opacities identified by Dr. Miller and Dr. Cappiello are due to a disease process other than pneumoconiosis.

I find that the preponderance of the evidence clearly shows that the Claimant has established that Mr. M. had a condition that showed up on x-ray as a one centimeter or greater opacity in his lungs. As noted above, Dr. Cappiello and Dr. Miller found either category A or B opacities on a total of four x-rays. The physicians who provided narrative x-ray readings also documented enlarging opacities or densities in Mr. M.'s upper lungs, consistent with conglomerate masses of pneumoconiosis. Dr. Scott and Dr. Wheeler did not designate category A or B opacities, but they described coarse infiltrates in both upper lungs,[5] bilateral pleural fibrosis in the apices, and interstitial infiltrates or fibrosis in the mid and lower lungs. In his review of Dr. Cappiello's and Dr. Miller's x-ray readings, Dr. Wheeler took issue with what he viewed as conflicting statements, but he did not discuss the designations of category A or B opacities. He indicated that only coal workers' pneumoconiosis and silicosis cause large opacities on x-rays, and he did not see these conditions. But he did not discuss the issue of whether the x-rays documented the presence of a large opacity. Dr. Scott, in reviewing Dr. Miller's and Dr. Cappiello's x-ray readings, seemed to agree that there was a process that showed up on x-ray as areas of opacification, but he felt that they did not represent the large opacities of silicosis.

[5] According to Dr. Spagnolo, an "infiltrate" is basically an irregular density on an x-ray, not necessarily specific for any particular lung disease.

Based on the designations by Dr. Cappiello and Dr. Miller, which are supported by the narrative x-ray readings, and which are not refuted by Dr. Scott or Dr. Wheeler,[6] I find that Mrs. M. has established that Mr. M. had a condition that showed up as a one centimeter or greater opacity in his lungs. I find that the Claimant has established the presence of an opacity measuring at least one centimeter in diameter as required by the plain language of 30 U.S.C. § 921(c)(3)(A).

*2. Etiology of the Opacity/ Mass*

In addition to establishing the existence of a one centimeter or greater opacity, § 718.304 requires that the etiology of these opacities be coal-dust related. Under *Scarbro*, once Mrs. M. establishes this etiology, the Employer must provide evidence that affirmatively shows the opacities are not there or that they are from a disease process other than complicated pneumoconiosis. Here, Dr. Miller and Dr. Cappiello concluded that Mr. M. had pneumoconiosis, and that the large opacities were due to pneumoconiosis

Dr. Wheeler noted minimal ill defined diffuse mixed linear and irregular interstitial infiltrates or fibrosis in both lungs, and focal infiltrates or fibrosis in the upper lobes on Mr. M.'s August 29, 1996 x-ray. He attributed these findings to inflammatory disease (autoimmune disease or usual interstitial pneumonitis), more likely than cancer. On Mr. M.'s September 16, 2002 x-ray, Dr. Wheeler again found coarse infiltrates or fibrosis, this time moderate, in the upper lobes, with pleural fibrosis near the right scapula more than the left, which he felt was compatible with inflammatory disease more likely than cancer. On reviewing Mr. M.'s March 4, 2003 x-ray, Dr. Wheeler noted moderate coarse infiltrates or fibrosis in the upper lobes, which he felt was compatible with inflammatory disease more likely than cancer. Finally, in his latest report, Dr. Wheeler attributed the "extensive infiltrates" on the x-rays to inflammatory disease such as histoplasmosis.

Dr. Scott noted bilateral diffuse interstitial and alveolar infiltrates on Mr. M.'s August 3, 2001 x-ray, which he felt were possible tuberculosis superimposed on non-specific linear fibrosis. On his review of Mr. M.'s March 4, 2003 x-ray, Dr. Scott noted bilateral infiltrates, with alveolar and interstitial components, and pleural reaction in the lateral right upper lung. He felt that these changes could be tuberculosis superimposed on a previous non-specific fibrosis.

Dr. Wheeler made it clear that he believes that complicated pneumoconiosis cannot be diagnosed by x-ray alone, and that an "exact diagnosis" usually requires open lung biopsy, transbronchial biopsy, or convincing microbiology. He stated that the ILO classification system was never intended to be used for making clinical judgments. However, under the statutory scheme, a claimant is not required to undergo invasive procedures to provide tissue confirmation that he has complicated pneumoconiosis. The condition referred to in the statute is not synonymous with the medical or clinical diagnosis of complicated pneumoconiosis. I find that Dr. Wheeler's opinions are based on assumptions that are contrary to the Act.

[6] Dr. Spagnolo did not review any x-ray films.

Finally, I find the opinions of Dr. Wheeler and Dr. Scott to be speculative regarding the etiology of the opacities identified by Dr. Miller and Dr. Cappiello. Thus, I find that they merely speculated that the extensive areas of infiltrates and fibrosis in Mr. M.'s lungs were attributable to another disease process, without substantiation or corroboration. Thus, there is no evidence in the record that Mr. M. has ever suffered from or been exposed to tuberculosis or histoplasmosis; nor is there any evidence in the record to suggest that he suffered from cancer or any autoimmune disorder.

Although Dr. Spagnolo did not review any x-ray films, in discussing Dr. Guadiano's diagnosis of progressive massive fibrosis or complicated pneumoconiosis, he indicated that there was no report of any large category opacities in the x-ray reports he reviewed. But he was not provided with the reports by Dr. Miller and Dr. Cappiello, and thus his opinions do not add anything to the determination of whether the large opacities that they identified were due to pneumoconiosis.

I have evaluated the x-ray evidence and find that the Claimant satisfied her burden to prove that Mr. M. suffered from the statutorily defined condition referred to as complicated pneumoconiosis. Thus, the Claimant has established that Mr. M. had a condition that showed up on x-ray as a Category A or B opacity. The Employer has failed to provide x-ray evidence affirmatively showing that the opacities or not there, or that they are due to a process other than pneumoconiosis.

*Weighing All Evidence Together*

Upon reviewing all of the evidence together, I find that the Claimant has established that Mr. M. suffered from the statutory condition referred to as complicated pneumoconiosis, and thus she is entitled to the irrebuttable presumption that Mr. M.'s death was due to pneumoconiosis. I find that the preponderance of the persuasive x-ray evidence establishes that Mr. M. had a condition that resulted in the presence of a large opacity on x-ray, due to his twenty eight years of occupational exposure to coal dust.

But the Employer has not offered affirmative evidence that the large opacity was due to something other than exposure to coal dust. Indeed, the record contains no evidence of exposure to causative agents other than coal dust, such as asbestos or tuberculosis. Nor are there any treatment records indicating that Mr. M. was ever diagnosed with or treated for tuberculosis, granulomatous, or any other pulmonary impairment that would produce opacities on an x-ray. Thus, I find that the preponderance of the evidence points to coal dust exposure as the etiology for Mr. M.'s radiographic abnormalities.

I find that Mrs. M. has established that she is entitled to the irrebuttable presumption that Mr. M.'s death was due to pneumoconiosis, pursuant to § 718.304.

I also find that Mrs. M. has established that Mr. M.'s death was due to pneumoconiosis under § 718.205(c)(2).[7] There are no medical records or other evidence to indicate the circumstances immediately surrounding Mr. M.'s death. Mr. M. apparently died at home, and was brought to the hospital by ambulance. Dr. Farooq's "Death Note," dated May 12, 2003, reflects that Mr. M. was treated by a paramedic, and that he died in his bed. Dr. Farooq completed Mr. M.'s death certificate, recording chronic obstructive pulmonary disease as the immediate cause of death, with pneumoconiosis and anemia as the underlying causes (DX 6).

Dr. Farooq was one of Mr. M.'s treating physicians, who saw him frequently in the last year of his life for his pulmonary problems, including chronic obstructive pulmonary disease and pneumoconiosis. Mr. M. visited the clinic on April 1, 2003, a little more than a month before his death; a notation by Dr. Nawaz indicates that Mr. M.'s pulmonary function tests were suggestive of severe obstructive disease that did not clearly improve with bronchodilators. Mrs. M. confirmed that Mr. M. had serious respiratory problems, and that he was on constant oxygen. Other than his breathing problems, Mr. M's medical records do not reflect any other significant medical problems, which was also confirmed by Mrs. M.[8]

Because Mr. M. died a "quick death" at home, and there was no report of any observation of breathing problems, Dr. Hippensteel speculated that his death was "most likely" due to arrhythmia, which would not produce symptoms of exacerbation of breathing problems that would precipitate a trip to the emergency room.[9] Dr. Hippensteel acknowledged that there was no evidence that Mr. M. had ever had a heart attack, or coronary artery disease, or even a workup for coronary artery disease. According to Dr. Hippensteel, the less serious types of arrhythmia can be controlled with medication, but there is no indication that Mr. M. took any such medication. Indeed, Dr. Chang's few notations of arrhythmia indicate that it was chronic stable, and asymptomatic. Dr. Hippensteel surmised, however, that Mr. M. had the more serious type of arrhythmia, that is, the ventricular type, which can cause sudden death. But this is not documented by Mr. M.'s medical records, nor did Dr. Hippensteel indicate how he arrived at this diagnosis. According to his wife, Mr. M. did not suffer from heart problems, hypertension, diabetes, or asthma. Nor do his medical records reflect a history of arrhythmias.

Dr. Hippensteel speculated that Mr. M.'s allergic rhinitis meant that he also had allergies that affected his lower airway, despite the fact that he was never diagnosed with asthma. He further speculated that this could have caused an obstructive disease resulting in intermittent exacerbations, such as those experienced by Mr. M. Dr. Chang's records include a few notations of allergic rhinitis, but they also clearly reflect that Mr. M.'s main problem was his severe and progressively worsening COPD. And setting aside the fact that the record does not reflect that Mr. M. was ever diagnosed with asthma, Dr. Hippensteel's suppositions ignore the fact that Mr. M.'s underlying respiratory condition was constant, with frequent trips to the hospital; as his wife testified, he was on constant oxygen, and unable to get around.

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[7] In making this finding, I rely on my conclusion that Mrs. M. has established that Mr. M. had pneumoconiosis under § 718.304; as Mr. M. had 28 years of coal mine employment, Mrs. M. is entitled to the statutory presumption, which has not been rebutted, that his pneumoconiosis was due to his coal mine employment.

[8] In his closing brief, Claimant's counsel refers to a report by Dr. Dominic Gaziano dated April 1, 2004. However, there is no such report in the record.

[9] It appears that Dr. Hippensteel assumed that there were no such symptoms of exacerbation of breathing symptoms. As no one was present when Mr. M. died, however, it is not clear how Dr. Hippensteel knew this.

I find that Dr. Hippensteel's conclusions are much too speculative to constitute persuasive evidence that pneumoconiosis played no part in Mr. M.'s death. There is nothing in the record to indicate that Mr. M. was ever diagnosed with or treated for any type of heart condition. And even if I were to accept the fact that he suffered from arrhythmia at some time in the past, that does not lead to the conclusion that his sudden death must have been due to an arrhythmia.

Dr. Hippensteel's attempt to attribute all of Mr. M.'s respiratory problems to asthma is also unpersuasive. Dr. Hippensteel speculated that because Mr. M. had symptoms of allergic rhinitis in the past, it was also likely that he suffered from asthma, which causes intermittent exacerbations of respiratory symptoms. He felt that this fit with Mr. M.'s pattern of frequent visits to the hospital for exacerbation of breathing problems. But even if Mr. M. had a component of asthma, which his physicians for some reason did not document, that does not preclude the co-existence of pneumoconiosis and COPD.

Again, I find that Dr. Hippensteel's opinions are speculative, and unsupported, even contradicted by, the objective medical evidence. I do not accord them any weight on this issue.

Dr. Spagnolo also advanced several possible causes for Mr. M.'s sudden death at home. Thus, he felt that Mr. M. "probably" had a cardiac arrhythmia, irregular heart rate, or acute myocardial infarction. He did not explain why he attributed Mr. M.'s death to these conditions, or reconcile them with the lack of any documentation of heart disease in Mr. M.'s medical records. Dr. Spagnolo also speculated that because Mr. M. had recurrent pneumonias, he may have died of a massive aspiration pneumonia. I find that Dr. Spagnolo's opinions on the possible causes of Mr. M.'s death are entirely speculative, and entitled to no weight.

As one of Mr. M.'s treating physicians, Dr. Farooq was familiar with his medical condition, as well as his x-rays, which Dr. Younis reported as early as August 3, 2001 showed diffuse changes of occupational lung diseases, and enlarging opacities that favored conglomerate masses of pneumoconiosis. Numerous x-rays up to March 2003 were performed at the Clinic, with similar and indeed progressive changes. At the time of his death, Mr. M. was being treated for his severe respiratory problems. I find that Dr. Farooq's conclusion that Mr. M.'s death was the immediate result of his COPD, with pneumoconiosis and anemia being underlying causes, is well-reasoned, and documented by Mr. M.'s medical records; I accord it determinative weight.

Accordingly, I find that the Claimant has established by a preponderance of the medical evidence that Mr. M.'s death was due to pneumoconiosis, pursuant to § 718.205(c)(2).

## CONCLUSION

Based on the foregoing, I find that the Claimant has established that Mr. M. had pneumoconiosis as a result of his coal mine employment, and that his death was due, at least in part, to his pneumoconiosis. Accordingly, she is entitled to benefits under the Act in connection with her survivor's claim.

## ORDER

Based on the foregoing, IT IS HEREBY ORDERED that the claim of M. F. M. for benefits under the Black Lung Benefits Act is granted.

IT IS FURTHER ORDERED that the Employer, Sewell Coal Company, shall pay to M. F. M. all benefits to which she is entitled under the Act.

SO ORDERED.

A

LINDA S. CHAPMAN
Administrative Law Judge

## ATTORNEY'S FEES

An application by Claimant's attorney for approval of a fee has not been received. Thirty days is hereby allowed to Claimant's counsel for submission of such an application. A service sheet showing that service has been made upon all the parties, including the claimant, must accompany the application. The parties have ten days following receipt of any such application within which to file any objections. The Act prohibits the charging of a fee in the absence of an approved application.

**NOTICE OF APPEAL RIGHTS:** If you are dissatisfied with the administrative law judge's decision, you may file an appeal with the Benefits Review Board ("Board"). To be timely, your appeal must be filed with the Board within thirty (30) days from the date on which the administrative law judge's decision is filed with the district director's office. *See* 20 C.F.R. §§ 725.458 and 725.459. The address of the Board is: Benefits Review Board, U.S. Department of Labor, P.O. Box 37601, Washington, DC 20013-7601. Your appeal is considered filed on the date it is received in the Office of the Clerk of the Board, unless the appeal is sent by mail and the Board determines that the U.S. Postal Service postmark, or other reliable evidence establishing the mailing date, may be used. *See* 20 C.F.R. § 802.207. Once an appeal is filed, all inquiries and correspondence should be directed to the Board.

After receipt of an appeal, the Board will issue a notice to all parties acknowledging receipt of the appeal and advising them as to any further action needed.

At the time you file an appeal with the Board, you must also send a copy of the appeal letter to Donald S. Shire, Associate Solicitor, Black Lung and Longshore Legal Services, U.S.

Department of Labor, 200 Constitution Ave., NW, Room N-2117, Washington, DC 20210. *See* 20 C.F.R. § 725.481.

If an appeal is not timely filed with the Board, the administrative law judge's decision becomes the final order of the Secretary of Labor pursuant to 20 C.F.R. § 725.479(a).